IN THE CLAIMS

Please cancel Claims 31-32 without prejudice.

Please amend Claim 1 as follows:

1. (Currently Amended) A method for reducing cardiac dysfunctions in a human in need thereof wherein the cardiac dysfunction is due to a pathological excess of norepinephrine release, the method comprising a administering to the human an effective amount of a selective histamine H_3 receptor agonist.

2. (Original) The method according to claim 1, wherein the cardiac dysfunction is associated with myocardial ischemia or myocardial infarction.

3. (Original) The method according to claim 1, wherein the cardiac dysfunction is arrhythmia, fibrillation, platelet activation and aggregation, thrombus formation, coronary spasm, sudden cardiac death or cardiac failure.

4. (Original) The method according to claim 1, wherein the selective histamine H_3 receptor agonist is R-(α)-methylhistamine, imetit, immepip, immepyr, 4-(1H-4-imidazolylmethylene)1-methylpiperidine, S-α-chloromethylhistamine, cyclopropyl-histamine, SKF 91606, Sch 50971, VUF 4864.

5. (Original) The method according to claim 1, wherein the selective histamine H_3 receptor agonist is administered after the onset of myocardial ischemia and/or myocardial infarction.

6. (Original) The method according to claim 1, wherein the selective histamine H_3 receptor agonist does not act on the central nervous system.

7. (Original) The method according to claim 1, wherein the selective histamine H_3 receptor agonist does not cross the blood brain barrier.

8. (Original) The method according to claim 1, wherein the histamine H_3 receptor is on a cardiac sympathetic nerve ending.

9. (Original) The method according to claim 1, wherein the histamine H_3 receptor agonist reduces norepinephrine release from a cardiac sympathetic nerve ending.

10. (Original) The method according to claim 1, wherein the reduction in norepinephrine release is specifically antagonized by an H_3R antagonist.

11. (Original) The method according to claim 1, wherein the H_3R antagonist is Thioperamide or Clobenpropit.

12. (Original) The method according to claim 1, wherein the histamine H_3 receptor agonist inhibits the Na^+/H^+ exchanger.

13. (Original) The method according to claim 12, wherein the histamine H_3 receptor agonist inhibits the Na^+/H^+ exchanger on a cardiac sympathetic nerve ending.

14. (Original) The method according to claim 1, wherein the histamine H_3 receptor agonist modulates the concentration of intracellular sodium.

15. (Original) The method according to claim 1, wherein the histamine H_3 receptor agonist modulates the concentration of intracellular calcium.

16. (Original) The method according to claim 15, wherein the histamine H_3 receptor agonist modulates the concentration of intracellular calcium by inhibiting the activity of an N-type Ca^{2+} channel.

17. (Original) The method according to claim 1, wherein the histamine H_3 receptor agonist is delivered in combination with at least one other agent in the treatment of cardiac dysfunction.

18. (Original) The method according to claim 17, wherein the other agent is one or more of the following: a β-blocker, a Ca^{++}-channel blocker, an anti-arrhythmic, an ACE inhibitor and an angiotensin receptor antagonist.

19. (Original) A method for inhibiting the Na^+/H^+ exchanger in a human having a cardiac dysfunction, the method comprising administering to the human an effective amount of a selective histamine H_3 receptor agonist.

20. (Original) The method according to claim 19, wherein the cardiac dysfunction is myocardial ischemia or myocardial infarction.

21. (Original) The method according to claim 19, wherein the cardiac dysfunction is arrhythmia, fibrillation, platelet activation and aggregation, thrombus formation, coronary spasm, sudden cardiac death or cardiac failure.

22. (Original) The method according to claim 19, wherein the selective histamine H_3 receptor agonist is R-(α)-methylhistamine, imetit, immepip, SKF 91606 or Sch 50971.

23. (Original) The method according to claim 19, wherein the selective histamine H_3 receptor agonist is administered after the onset of myocardial ischemia and/or myocardial infarction.

24. (Original) The method according to claim 19, wherein the selective histamine H_3 receptor agonist does not act on the central nervous system.

25. (Original) The method according to claim 19, wherein the selective histamine H_3 receptor agonist does not cross the blood brain barrier.

26. (Original) The method according to claim 19, wherein the histamine H_3 receptor is on a cardiac sympathetic nerve ending.

27. (Original) The method according to claim 19, wherein the histamine H_3 receptor agonist inhibits norepinephrine release from cardiac sympathetic nerve endings.

28. (Original) The method according to claim 19, wherein the histamine H_3 receptor agonist modulates the concentration of intracellular sodium.

29. (Original) The method according to claim 19, wherein the histamine H_3 receptor agonist is delivered in combination with at least one other agent in the treatment of cardiac dysfunction.

30. (Original) The method according to claim 19, wherein the other agent is one or more of the following: a β-blocker, a Ca^{2+}-channel blocker, an anti-arrhythmic, an ACE inhibitor and an angiotensin receptor antagonist.

Claims 31-32 (Cancelled).